Chapter 19

Osteological and Dental Pathology

As we have learned in the preceding chapters, bones can and do vary greatly between individuals. This variation, as introduced in Chapter 3, stems from four main sources: age, sex, ancestry, and idiosyncrasy. Idiosyncratic variation includes nonpathological variation between individuals (for instance, variation due to stature or to slight differences in developmental timing) as well as nonmetric, nonpathological variations that are essential in reconstructing various biological dimensions of former human populations (see Chapter 21).

This chapter introduces a variety of biological processes that can result in skeletal modifications before death (**antemortem changes**). These processes can be biological (*e.g.,* disease, genetic disorders), environmental (*e.g.,* trauma, nutritional deficiencies), cultural (*e.g.,* intentional deformation, surgery), or a combination of these. Cultural practices that take place just before or after death (**perimortem modifications**) are also examined. While these topics are all included, the discussion of skeletal modifications in this chapter focuses on pathological conditions. Chapter 20 examines some additional modifications, but there we concentrate on changes to the bone that occur after the death of the individual (**postmortem changes**). The most critical step in diagnosing and recording paleopathological conditions is the recognition of true bone abnormalities, as opposed to those within the normal range of variation in immature and adult healthy individuals. It is also vital to be able to distinguish between taphonomic and pathological alterations.

Paleoepidemiology is the study of factors affecting health and the risks of disease in past populations. Paleoepidemiology is concerned with only certain aspects of diseases: the cause(s), frequency, distribution, and transmission of a disease, as well as certain factors associated with the risks of contracting diseases. Waldron (1994) characterized paleoepidemiology as the attempt "to count the dead and their pathological signs in archaeological series, in order to reconstruct the spatial, temporal and social distribution of health and disease in past populations based on biocultural models."

Paleopathology was defined by one of its pioneers as "the science of the diseases which can be demonstrated in human and animal remains of ancient times" (Ruffer, 1913). Whereas paleoepidemiology examines the ways in which diseases interact with *populations,* paleopathology is primarily concerned with the ways in which diseases interact with *individuals.* The scope of paleopathology has grown considerably since its earliest applications in archaeology (Ruffer, 1913) and paleontology (Shufeldt, 1893). In addition to diseases, paleopathologists also study trauma, antemortem cultural modifications of the skeleton and teeth, degenerative conditions, age-related bone loss, occupational indicators, and nutritional deficiencies and other signs of dietary stress. Paleopathology has had its own professional society for nearly 40 years (the Paleopathology Association), and will have its own international scientific journal, the *International Journal of Paleopathology,* in 2011.

The primary data of paleopathology are fossilized or skeletal remains, preserved soft tissues, and molecular evidence (see Chapter 22). Bones and teeth can be records of events during the life of an individual, including trauma and disease. Indeed, dramatic insights into the health of individuals, and of populations, may be gained from studies in osteological pathology. As Miller and colleagues (1996) note, the objectives of paleopathological research include diagnosing specific diseases in individual skeletal remains and examining the populational and evolutionary effects of these interactions between humans and the diseases that afflict them.

Pathological changes observable in osteological materials result from an imbalance in the normal equilibrium of bone resorption and formation, or from growth-related disorders. This imbalance can arise as a result of many factors, including mechanical stress, changes in blood supply, inflammation of soft tissues, changes brought about by infectious diseases, hormonal, nutritional and metabolic upsets, and tumors (Mensforth et al., 1978). Diagnosing the exact cause of an observed skeletal pathology is, however, not always possible. For example, an individual's growth may be interrupted by a range of factors that include infectious disease, starvation, and/or trauma. This growth arrest may lead to the formation of **Harris lines** in the long bones, lines of increased bone density that represent the position of the growth plate at the time of insult to the organism. The lines are visible radiographically, or in cross section, and may be used to estimate the age at which the individual was stressed.

Radiographic assessment is an essential component of describing and diagnosing disease in skeletal remains. Observations on different individuals may be combined to estimate the degree to which the population was stressed. From the Harris lines themselves, however, only general statements about the variety of possible stressors (including disease and diet) may be made (Maat, 1984; Hummert and Van Gerven, 1985). Furthermore, the lines may be removed by bone remodeling after they form. These Harris lines illustrate yet another problem common to comparative work in paleopathology. Macchiarelli and colleagues (1994) have shown that **interobserver error** may be high in radiographic interpretations, and even the scoring of the same radiographs by the same individual on different occasions (**intraobserver error**) can result in reported differences. Waldron and Rogers (1991) expressed similar concerns based on a study of interobserver variation in coding osteoarthritis in skeletal remains.

19.1 Description and Diagnosis

There are two stages involved in a paleopathological examination: describing the pathological manifestations and diagnosing the cause of these manifestations. Whereas historically an emphasis was placed on diagnosis (*e.g.*, Steinbock, 1976), modern practice regards the full, accurate, and unambiguous description of pathological conditions as being of primary importance (Rose et al., 1991; Buikstra and Ubelaker, 1994).

Books by Steinbock (1976), Ortner and Putschar (1981), Ortner and Aufderheide (1991), Aufderheide and Rodríguez-Martín (1998), Ortner (2003), Mann and Hunt (2005), Roberts and Manchester (1995, 2007), Pinhasi and Mays (2008), and Waldron (2009) provide excellent guides to osteological pathology, and Lewis (2000) provides a status report on paleopathology for nonadults. Additional illustrations and techniques are found in Buikstra and Ubelaker (1994) and Lovell (2000). Hillson (2008) provides a review of dental pathology. The reader is urged to consult these sources when working with potentially pathological human skeletal remains. This chapter is intended to supplement these texts and is organized accordingly. It is designed to provide the reader with an introduction to the kinds of dental and osteological pathology encountered most commonly in work with prehistoric skeletal remains. All illustrated examples of osteological pathology are drawn from the Phoebe A. Hearst Museum of Anthropology, most of them from archaeological contexts. After examining 5,000 individuals in this collection, Richards and Antón (1987) note that over one-fifth showed anomalous development or the effects of pathological processes. Their data show that degenerative joint disease, periodontal disease, fracture, and osteomyelitis are the leading changes observed in this mostly prehistoric Californian collection.

19.1.1 Description

The first step in any paleopathological analysis is to establish the envelope of what is normal in size, shape, and topography for a healthy human's skeleton. When skeletal remains fall outside that envelope, pathology is one possible explanation. The basic steps are **description** and **diagnosis**. Diagnosis will rarely approach what is possible in the clinical setting because bone usually responds to insults by either resorbing or depositing, and it may therefore easily respond to different diseases in very similar ways. Description is the most important step in paleopathological work because even if the subsequent diagnosis is incorrect, other workers can come along later and amend or modify the diagnosis.

In any description, identify the nature and distribution of anomalies across the skeleton and observe the pattern of distribution of these anomalies across the population. These anomalies can take the form of lesions or of changes in size or shape. Note the distribution of all lesions, whether single, multiple, diffuse, or concentrated. Note whether bone is eaten away (**lytic**) or deposited (**blastic**). Check all bones of the skeleton for further evidence, and verify that the alteration is not taphonomic (Chapter 20), a nonpathological anatomical variant, or some other example of "**pseudopathology**."

Pathological conditions have been organized in a number of different ways, according to the type of work being done (*e.g.,* data gathering vs. analysis), the type of data examined (*e.g.,* gross anatomical vs. histological vs. genetic), the subset of data being examined (*e.g.,* skeletal manifestations), and/or the conceptual framework into which the data are placed (*e.g.,* evolutionary vs. etiological vs. symptomatological). The World Health Organization's most recent *International Statistical Classification of Diseases and Related Health Problems* (World Health Organization, 2007) is the comprehensive standard for pathology. Paleopathologists usually use much simpler classification schemes. Two particular classifications are of special interest to paleopathologists — one to guide description and data collection, and the other to assist in diagnosis.

Buikstra and Ubelaker (1994: 112–115), building on the work of Rose et al. (1991), advocate the division of paleopathological changes into nine categories for the purpose of data collection: (1) abnormalities of shape, (2) abnormalities of size, (3) bone loss, (4) abnormal bone formation, (5) fractures and dislocations, (6) porotic hyperostosis/cribra orbitalia, (7) vertebral pathology, (8)

1.0.0 General abnormality of shape (long bone)
- 1.1.0 Bowed (abnormal curvature)
- 1.2.0 Angulated
- 1.3.0 External outline altered
  - 1.3.1 Flaring metaphyses
  - 1.3.2 Uniformly abnormally wide
  - 1.3.3 Fusiform (spindle) shape
  - 1.3.4 Other (see narrative)
- 1.4.0 Degree of shape abnormality
  - 1.4.1 Barely discernible
  - 1.4.2 Clearly discernible

1.0.0 General abnormality of shape (skull)
- 1.5.0 Craniosynostosis, suture
  - 1.5.1 Metopic
  - 1.5.2 Coronal
  - 1.5.3 Sagittal
  - 1.5.4 Lambdoid
  - 1.5.5 Other (see narrative)
- 1.6.0 Craniosynostosis, completeness
  - 1.6.1 Partial
  - 1.6.2 Complete

1.0.0 General abnormality of shape (vertebrae)
- 1.7.0 Type
  - 1.7.1 Kyphosis (ant.-post.)
  - 1.7.2 Scoliosis, left
  - 1.7.3 Scoliosis, right
- 1.8.0 Form
  - 1.8.1 Angular
  - 1.8.2 Gradual change in body height
- 1.9.0 Ankylosis
  - 1.9.1 Absent
  - 1.9.2 Present

Table 19.1 **Examples of paleopathology data collection codes.** From Buikstra & Ubelaker (1994).

arthritis, and (9) miscellaneous conditions. For each of these categories, Buikstra and Ubelaker present a number of clearly defined generic and specific findings. Using this system, every pathological feature is recorded as a combination of one or more observation codes (see Table 19.1) with one or more bone and side codes, with codes for aspect and section used as needed. It is important to note that Buikstra and Ubelaker's system is intended only for description, not for diagnosis.

19.1.2 Diagnosis

When trying to formulate a diagnosis, the osteologist is at a decided disadvantage compared to the forensic pathologist or clinician. Whereas a clinician can monitor progress of a disease in a patient, the paleopathologist is limited to the static appearance of the skeleton at the time of death. Furthermore, in the majority of paleopathology cases, diagnosis is necessarily based on gross appearance and radiology. In contrast, the clinician diagnosing disease in a living patient can assess patient history, soft tissue, chemistry, pathogens, and pain. It follows that paleopathological diagnoses based on skeletal lesions will rarely have the precision routinely encountered in clinical settings. Developments in genetics, biochemistry, and molecular biology, however, are opening new doors for the paleopathologist.

Techniques and methods available for paleopathological diagnosis include (in addition to gross anatomy) histology, radiography, computed tomography, microradiography, scanning electron micrography, chemical analysis, serology, and genetic sequencing.

A number of diseases are genetic in nature and can potentially be detected in past populations using DNA analysis. Disease processes characterized by long-term infection by substantial densities of viral or bacterial pathogens might also be detected through recovery of the DNA of the pathogen. As yet, very few studies of paleopathology have utilized DNA analysis, in large part due to the difficulty of avoiding contaminant DNA from living humans. Whereas the DNA of ancient humans is difficult to isolate, it is a relatively straight-forward process to isolate the DNA of infectious pathogens, so most genetic analyses have focused there. Most work to date has been done on tuberculosis, amplifying the DNA of *Mycobacterium tuberculosis* (Braun et al., 1998; Donoghue et al., 1998; Fletcher et al., 2003; Hass et al., 2000; Mays et al., 2001; Spigelman et al., 2002), but leprosy (Haas et al., 2000; Montiel et al., 2003; Taylor et al., 2000), malaria (Sallares and Gomzi, 2001), plague (Drancourt et al., 1998; Raoult et al., 2000), and syphilis (Kolman et al., 1999) have also received attention. Roberts and Ingham (2008) take a critical look at DNA analysis in paleopathology, finding that a majority of the genetic studies done did not follow established standards for such work. Bouwman and Brown (2005) also express concern over the lack of rigorous validation of results in some DNA analyses in paleopathology.

Unfortunately for paleopathologists, few diseases leave signatures of any kind on the human skeleton, and those that do may cause very similar skeletal reactions. The only real advantage that the osteologist has in studying pathology is the ability to study the entire skeleton at once, without soft tissue cover. For these reasons, Steinbock (1976) suggested that the most rational approach to differential diagnosis in human skeletal pathology is to state the most likely diagnosis followed by a list of possible alternatives in order of decreasing likelihood.

As Miller et al. (1996) note, there are two major impediments to paleopathological diagnoses. First, there is a paucity of well-documented, clinically diagnosed skeletal samples to use as controls against which unknown skeletal samples (forensic, archaeological, or paleontological) may be compared. The second problem lies with the difficulty in finding skeletal abnormalities, or patterns of abnormalities, that are unique to individual disease categories. Miller et al. (1996) suggest that paleopathologies be diagnosed and classified in one of seven categories: (1) anomaly, (2) trauma repair, (3) inflammatory/immune, (4) circulatory (vascular), (5) metabolic, (6) neuromechanical, or (7) neoplastic (cancers). In a series of blind tests, however, these authors found that even trained specialists working on skeletal remains only achieved accuracies of about 43% at this broad diagnostic level, while only about 30% of more specific diagnoses were accurate.

19.2 Skeletal Trauma

The most common pathology affecting the skeleton is degenerative change. Trauma occupies second place and affects the skeleton in several ways — fracturing or dislocating the bone, disrupting its blood or nerve supply, or artificially deforming it. For illustrations of trauma to osteological remains, see White (1992) and the FOROST visual metabase and other online resources (Appendix 3). Ortner (2003) divides skeletal trauma into four general types: (1) a partial to complete break in a bone, (2) an abnormal displacement or dislocation of a joint, (3) a disruption in nerve and/or blood supply, or (4) an artificially induced abnormal shape or contour of a bone. Skeletal trauma can be the result of an accident, interpersonal violence, cultural practice, or therapeutic treatment. Martin and Frayer (1997) give examples of how studies of osteological trauma have been employed to investigate violence and warfare in the past. Lovell (2008) provides a recent overview of research on skeletal trauma.

19.2.1 Fracture

Fractures of bones can occur for numerous reasons, most often as a result of sufficiently strong abnormal forces of tension, compression, torsion, bending, or shear applied to a bone. The terms used to describe fractures are equally wide-ranging, especially in the field of medicine. Commonly recognized fracture types and terms are mentioned here. Exploring the myriad of different types of fractures and their causes is beyond the scope of this work. Refer to Table 19.2 for a list of common fractures and brief descriptions of the trauma.

A fracture in which broken ends of a bone become separated is called a **complete fracture**. If the discontinuity does not bifurcate the bone, it is an **infraction**, or **incomplete fracture**. In a **compound** (or **open**) **fracture**, part of the broken bone perforates the skin. The skin remains intact in a **simple** (or **closed**) **fracture**. Additional types of fractures related to these four main categories are listed in Table 19.2.

Figure 19.1 **Healed fracture.** This left clavicle shows a postmortem fracture (the light-colored bone around the crack visible in this inferior view) and a more medial antemortem fracture that has healed. The original shaft surfaces are joined by a bony callus. The radiograph shows that the medial end of this clavicle rotated counterclockwise relative to its lateral end, resulting in a dramatic misalignment of the fractured pieces during and after healing. Prehistoric, California. One-half natural size.

Table 19.2 **Useful terms for describing fractures**

Forces	
tension	stress that produces elongation, stretching
compression	pressed together, typically affecting the spine
torsion	twisting
bending	deformation from a normally straight condition, but not to the point of breaking
shearing	forced applied to the side of an immobilized bone segment
dynamic	sudden stress with power and velocity
static	slowly applied stress
narrow focus	stress applied at a point or line
wide focus	stress applied over a large area
Fractures (main categories)	
complete	a bone breaks into separate pieces
transverse	a clean, square break perpendicular to the bone's long axis
linear	a fracture along the bone's long axis.
oblique	clean break diagonal to the bone's long axis
displaced	broken ends are separated producing a fracture gap, often left at an angle from normal (angulation)
nondisplaced	a complete fracture with the broken bone remaining in its normal position and alignment
compressed	bone tissue collapses (common in vertebrae)
incomplete (infraction)	a crack, bend, or break with fragments remaining partially joined, but not a complete break (no separation)
greenstick	the bone bends on one side and breaks on the other like a bent green tree branch (most common in children)
torus (buckle) fracture	the bone bends producing a raised buckle on one side, but no break on the other side.
compound (open)	the break goes through the skin (and may recede back)
closed	the fracture does not pass through the skin
Other Fracture Types	
pathological	when bone is weakened or made brittle by disease (osteoporotic, infection, cancer, noncancerous tumor)
fatigue	when bone is exposed to [intermittent] stress over a long period.
stress	cracks in the bone caused by repeated strain and overuse
avulsion	small piece of bone detaches where ligaments and tendons attach
simple (single)	a single discontinuity along one line produces two bone segments
segmental	a long bone broken in two or more places
comminuted (multi-fragmentary)	the break produces several (three or more) pieces of bone or fragments
spiral	a torsion fracture caused by twisting
depressed	pushed in with fragments of bone depressed below the adjacent surface
diastatic	a widening or separation of the cranial bones at the sutures
basilar	a bone break at the base of the skull
hinge	a peeling or flap of bone, still attached at one side, caused by a sharp force across the cortical surface of bone
impacted/compacted	broken fragments are embedded into each other
cleft	a V-shaped notch caused by a near vertical force applied by a heavy instrument with a sharp edge
fracture lines:	
radiating	fracture lines spreading outward from an impact point where a force was applied
concentric (hoop)	fracture lines occurring in concentric rings around the area of applied force
LeFort fractures of the face	
I	separation of the alveolar part of the maxilla between the alveolar ridges and the frontal processes
II	separation of the mid-face from the rest of the cranium
III	the entire face is separated from the braincase
Types of Trauma	
blunt force (BFT)	injury due to a force with a wide area of impact
sharp	a compressing or shearing dynamic strike with a narrow focus
projectile	a combination of blunt and sharp characteristics
sawing	cutting bone with a toothed instrument
heat	discontinuities caused by the effects of heat on tissue
chemical	physical damage caused by toxic substances; immediate as in a chemical burns, or poisoning over time

Fractures of bones are often described by the features of the break itself: **comminuted** when there is shattering of bone; **compressed** when the bone is squeezed; or **depressed** when bone fragments are depressed below the adjacent surface. Pathological or metabolic conditions such as osteoporosis and cancer weaken bone and can lead to a **pathological fracture**. Several possible types of gross fracture in a bone may result from this abnormal stress (Figures 19.1 and 19.2).

Characteristics of bone trauma caused by a striking object vary depending on the type of bone involved and the features of the object. Fracture margins may reveal information regarding the nature of the force that caused the fracture. The result of a blunt object impacting bone is different from a fracture caused by a stabbing action with a sharp object. Some fractures are caused by forces applied in one location that radiate or become concentrated elsewhere. For example, diastatic fractures occur along cranial sutures and may be caused by stress dissipating across the skull due to forces applied at a different location.

Fracture healing is described in Chapter 3. Antemortem fracture may be differentiated from postmortem fracture only when a **callus**, the hard tissue formed at the site of a broken bone during the healing process, is present. All other fractures that occur at or around the time of death should be diagnosed as perimortem. Complete fracture healing can completely remove any gross signs of fracture, even in a radiograph. The rate of fracture repair depends on fragment alignment, the amount of movement at the site of fracture, and the health, age, diet, and blood supply

Figure 19.2 **Healed fracture.** *(Left)* This left radius has a healed midshaft fracture. The radiograph shows that the fracture offset is about equal to the width of the bone. The proximal part of the bone is offset posteriorly. Prehistoric, California. One-half natural size.

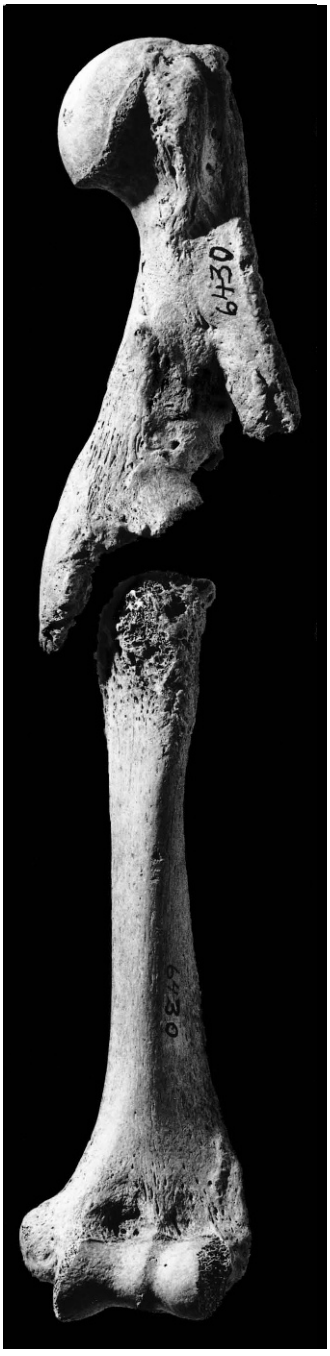

Figure 19.3 **Pseudarthrosis.** *(Right)* This left humerus was broken just above the midshaft. The fracture failed to heal (a nonunion), resulting in continued movement, which formed the false joint. Prehistoric, California. One-half natural size.

of the individual. Some fractures never heal because of continued movement at the broken surface. Nonunions develop most frequently in the appendicular skeleton. It is possible that a new "joint," or **pseudarthrosis**, will form at the fracture site (see Figure 19.3).

Trauma such as sword cuts or arrow perforations to bones constitute special kinds of fractures. Such wounds are capable of healing through the same processes described in Chapter 3. In any kind of fracture, adjacent bone is susceptible to subsequent pathological complications such as infection, tissue death, deformity, and arthritis brought on by the initial trauma.

The analysis of fractures at the populational level can be very informative in addressing questions of prehistoric behavior. For example, Lovejoy and Heiple (1981) assessed the Libben population and found that the overall fracture rate was high. The low incidence of fractures in children suggested that traumatic child abuse was not practiced. The results of their analysis also suggested that fracture risk was highest in the 10–25 and 45+ age categories and that the care of patients was enlightened and skillful among this Native American group. Andrushko et al. (2005) investigate the practice of trophy-taking by means of partial dismemberment. Jurmain et al. (2009) examine the skeletal evidence for interpersonal violence in prehistoric California, finding craniofacial trauma and projectile injury more reliable indicators than forearm injury.

19.2.2 Dislocation

In addition to causing bone fracture, trauma to the skeleton can also involve movement of joint participants out of contact with the simultaneous disruption of the joint capsule. If the bones participating in the joint remain dislocated, the result may be diagnosed osteologically (Figure 19.4). When the joint is dislocated, the articular cartilage cannot obtain nourishment from the synovial fluid, the cartilage disintegrates, and arthritic changes occur. Osteological manifestations of dislocation are usually confined to adults. The violent trauma necessary for dislocation usually separates the epiphyses in subadults, and slipped femoral epiphyses are common in juveniles. In the elderly, the more brittle bone usually gives way, fracturing prior to dislocation. The two joints most often displaying osteological manifestations of dislocation are the shoulder and hip joints.

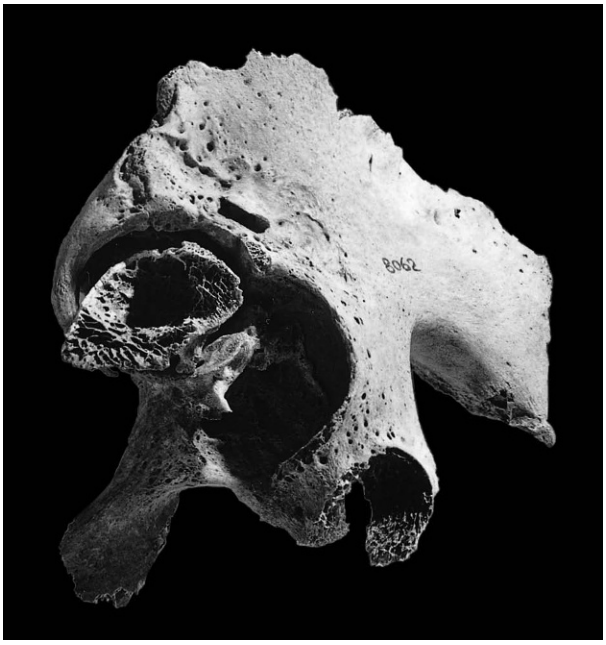

Figure 19.4 **Dislocation.** This left hip joint shows that the femoral head dislocated anterosuperiorly from its original place within the acetabulum. The cross section of the femoral neck is seen in the postexcavation break, which faces the viewer. Osteoarthritis secondary to the trauma is evident. Prehistoric, California. One-half natural size.

19.2.3 Vascular Disruption

When the blood supply to a bone is upset by trauma or other diseases, a variety of bony manifestations can occur, including death of bone tissue (**osteonecrosis**). See Section 19.4 for examples of pathologies that can result from vascular disruption.

19.2.4 Artificially Induced Shape Changes

In addition to accidental trauma, there are also several types of intentional trauma. Bone shapes can be modified by the application of long-term compressive forces, as with artificial cranial deformation and foot binding. Certain therapeutic procedures can also modify a bone's shape, as can some punitive measures.

a. **Artificial Deformation**. Fracture and dislocation described earlier result from sudden trauma to the skeleton, but sustained mechanical stresses can also modify the shape of a bone. Deformities of this kind are induced most often as a result of cultural practices such as cradleboarding, massaging, or binding the crania of infants. Another example is the foot-binding practiced by Chinese women of high status. The most common manifestations of artificial deformation of the skeleton are those of the cranium. Cultures around the world have, for cosmetic reasons, altered the shape of the adult head by placing abnormal pressures on the developing skull. Ortner and Putschar (1981) describe this practice by people on every continent except Australia, but Brown's (1981) work there suggests that the practice was continentally ubiquitous. Both cultural and biological information can come from the analysis of intentionally deformed crania. For example, Antón (1989)

Figure 19.5 **Artificial cranial deformation.** Circumferential deformation is produced by wrapping the rear of the cranial vault. Prehistoric, Peru. One-half natural size.

has used intentional anteroposterior and circumferential cranial vault deformation in a Peruvian sample to study the relationship between the cranial vault and the base in the development of the craniofacial complex. O'Loughlin (2004) notes the effect of cranial deformation increasing wormian bone number. Some examples of artificial cranial deformation are illustrated in Figures 19.5 and 19.6.

b. **Trephination**. Ortner and Putschar (1981) describe **trephination**, or **trepanation**, as perhaps the most remarkable trauma encountered by the paleopathologist (Figure 19.7).

Figure 19.6 **Artificial cranial deformation.** Anteroposterior deformation is caused by the application of pressure from behind. Prehistoric, Peru. One-half natural size.

Figure 19.7 **Trephination.** Three artificially produced holes are evident on this three-quarter view of a cranium. The hole closest to the parietal boss is nearly completely obliterated by healing. The other holes also show substantial bony healing, indicating that the individual survived the operations. Prehistoric, Peru. One-half natural size.

Written accounts documenting this practice extend to the ancient Greeks, but archaeological work has traced it even more deeply into the past. The practice is known from Europe, the Pacific, both Americas, Africa, and Asia. Several techniques have been used to make an artificial hole in the cranial vault, including scraping a patch of bone away, cutting a bone patch out by cutting grooves through the vault, and drilling small holes around the plug of bone to be removed. The practice was probably undertaken to yield relief from intracranial pressure (especially from compressive fractures of the skull vault) and to relieve headaches, cure mental illness, or let out evil spirits. The success rate for this prehistoric surgery, as judged by subsequent healing around the hole, was often surprisingly high. Some postmortem trephination was done to fashion amulets for the adornment of survivors. Arnott et al. (2003) provide an overview of trephination, and Andrushko and Verano (2008) examine trephination in ancient Peru.

c. **Amputation**. Evidence of amputation may be observed on the skeleton in the form of missing appendages or parts of appendages. Distinguishing antemortem from perimortem amputation, again, depends on the presence of healing or infection of the bone tissue at the point of trauma. Andrushko et al. (2005, 2010) investigate the practice of perimortem amputation and dismemberment in the context of trophy-taking.

19.3 Congenital Disorders

Congenital disorders result from developmental anomalies in, or damage to, a fetus. Causes of congenital disorders vary: genetic or chromosomal abnormalities, *in utero* environmental variables, and errors of morphogenesis are some of the possible factors.

19.3.1 Cranial Malformations

a. **Craniosynostosis**. Craniosynostosis is a general term that refers to the premature fusion of one or more cranial sutures. The resulting shape of the cranium depends on the sutures involved as well as the order in which the sutures fuse. There are several specific, named types of craniosynostosis. **Plagiocephaly** results from asymmetric suture closure and results in a "lopsided" appearance for the cranium. **Scaphocephaly** is the most common form of craniosynostosis (Aufderheide and Rodríguez-Martín, 1998), resulting from premature fusion of the sagittal suture.

b. **Microcephaly**. Microcephaly refers to an abnormally small neurocranium. In individuals with microcephaly, an abnormally low brain volume relaxes the need for a large endocranial space, resulting in a small neurocranium with a normal-size face. Richards (1985) describes a microcephalic child from prehistoric California.

c. **Hydrocephaly**. Hydrocephaly is a consequence of chronically increased intracranial pressure during childhood, resulting in a large, globular cranium, thin cranial vault bones, and bulging fontanelles. Richards and Antón (1991) describe a case of hydrocephaly from prehistoric California.

19.3.2 Vertebral Malformations

a. **Spina bifida**. When the two halves of the neural arch fail to fuse, the vertebral canal is left exposed, a condition called spina bifida. Spina bifida can occur at any level of the vertebral column, but is most common in the sacrum. Non-fusion of the lower sacral vertebrae is considered normal; only non-fusion of S-3 or any superior vertebra is considered spina bifida.

b. **Scoliosis**. Scoliosis refers to a lateral deviation of the vertebral column from the midsagittal plane. Scoliosis usually involves two abnormal curvatures, allowing the head to remain vertical and close to the midline.

c. **Kyphosis**. Kyphosis is an abnormal increase in the anterior curvature of the thoracic spine. Kyphosis can be primary (changes in the geometry of the intervertebral disks) or secondary (changes in the geometry of the vertebral bodies themselves). Kyphosis can occur as a result of any of a number of conditions, including osteoporosis, tuberculosis and other infectious diseases, multiple myeloma, osteomalacia, and acromegaly (Ortner, 2003).

19.3.3 Other Congenital Malformations

In addition to the cranial and vertebral malformations just listed, there are many other congenital conditions that affect the skeleton: absences of elements (aplasias), and incompletely developed elements (hypoplasias), clefts (*e.g.,* cleft palate, bipartite patella), abnormal geometries (*e.g.,* coxa vara), and supernumerary elements (*e.g.,* cervical ribs, polydactyly), are congenital conditions affecting the skeleton. Chapter 4 of Aufderheide and Rodríguez-Martín (1998) provides a good review of these and other congenital anomalies.

19.4 Circulatory Disorders

When the blood supply to a bone is disrupted, whether by trauma or other diseases, a variety of bony manifestations can occur. If the blood supply to a bone is gradually reduced, the bone will atrophy but will usually survive. If the blood supply is suddenly cut off, the affected bone tissue will become necrotic. **Osteochondroses** are joint diseases involving interruption of the blood supply (usually to an epiphysis), localized necrosis, and subsequent regrowth of the bone.

19.4.1 Osteochondritis Dissecans

In younger adults, localized trauma to long bone joint surfaces can result in the avulsion of small areas of subchondral bone. Osteochondritis dissecans is most commonly observed on the distal femur (especially the medial condyle), proximal talus, distal humerus (especially the capitulum), proximal ulna, humeral head, and patella. After the trauma, localized necrosis sets in, eventually causing the affected subchondral bone to separate. The resulting pit exposes the underlying trabecular bone, but a local sclerotic reaction eventually lines the pit with a new surface of bone.

19.4.2 Legg-Calvé-Perthes Disease

In Legg-Calvé-Perthes (LCP) disease, the blood supply to the femoral head epiphysis is disrupted. Whether this disruption is a result of trauma or of some other cause(s) is currently unknown (Wainwright and Catterall, 2010). While the femoral head atrophies due to necrosis, the femoral head's articular cartilage continues to grow, as its nutrition comes from synovial fluid, not the femoral head. Because of the necrosis, the femoral head becomes flattened and uneven at its margins. Once the blood supply is re-established, the femoral head begins to heal and grow, although usually maintaining its deformed shape. LCP disproportionately affects boys and Caucasians, and appears to be related to poor living conditions (Wainwright and Catterall, 2010). The onset of LCP is usually between 4 and 7 years, but the deformity persists through life.

19.5 Joint Diseases

Diseases of the joints are typically organized into two broad categories: bone-forming joint lesions (*e.g.,* osteophytes, syndesmophytes), and bone-eroding joint lesions (*e.g.,* eburnation) (Ortner, 2003). These categories, although sometimes useful, can be somewhat misleading, as there are types of bone-forming joint lesions that result in bone erosion and vice versa (Aufderheide and Rodríguez-Martín, 1998).

19.5.1 Osteoarthritis

Arthritis is the inflammation of a joint — a general inflammation that includes soft tissue effects. The inflammation can come as a result of trauma as well as of bone and joint infections (see Rogers et al. [1987] for a summary and classification). **Osteoarthritis**, the most common form of arthritis, is characterized by destruction of the articular cartilage in a joint and formation of adjacent bone, in the form of bony lipping and spur formation (**osteophytes**) around the edges of the joint. A better term for this phenomenon is **degenerative joint disease** (Figures 19.8–19.10). The causes of this disease are, for the most part, mechanical. The disease occurs mostly in load-bearing joints, particularly in the spine, the hip, and the knees. Osteoarthritis is an inherent part of the aging process. For a review of current knowledge about osteoarthritis, consult Epstein (1989), and for a review of prehistoric arthritis in the Americas, see Bridges (1992).

Osteoarthritis is usually classified as either primary, resulting from a combination of factors that include age, sex, hormones, mechanical stress, and genetic predisposition, or secondary, initiated by trauma or another cause such as the invasion of the joint by bacteria (**septic**, or **pyogenic arthritis** — often a complication of osteomyelitis). Studies of the patterning of osteoarthritic lesions of the skeleton at the individual and at the population level can shed light on prehistoric activity patterns. For example, Merbs (1983) was able to show that osteoarthritic changes seen in female Hudson Bay Inuit skeletons correlated with ethnographic accounts and archaeological evidence for scraping and cutting animal hides and sewing, whereas lesions seen in males correlated with harpoon throwing and kayak paddling.

A phenomenon often found associated with osteoarthritis is **eburnation**, the result of subchondral bone being exposed when cartilage is destroyed. Bone affected this way takes on a polished, ivory-like appearance (Figure 19.9). The projecting spicules of bone associated with osteoarthritis are called **osteophytes**. Nearly all individuals older than 60 years exhibit these arthritic features, especially in the lower thoracic and lumbar regions. Rogers et al. (1987, 1989) assess classifications of osteoarthritis, and Bridges (1993) notes that the scoring procedures used to record the disease seriously affect the results of any comparative study.

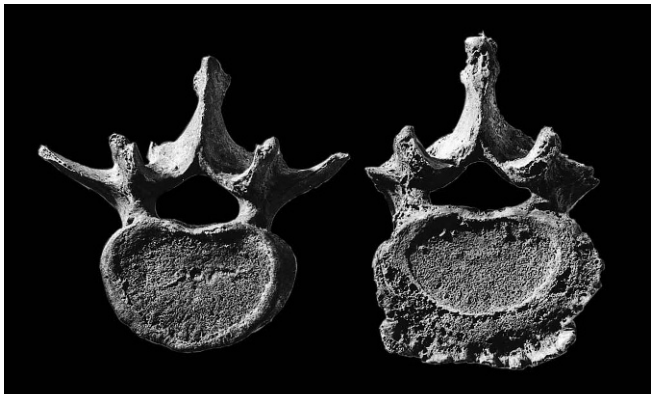

Figure 19.8 **Osteoarthritis.** The normal lumbar vertebra (left) lacks the osteophyte development seen on the anterior and lateral edges of the vertebra with degenerative arthritis (right). Prehistoric, California. One-half natural size.

Figure 19.9 **Osteoarthritis.** Eburnation and marginal lipping are evident on this left knee joint, seen here in posterolateral view and lit from the lower right to show detail. The eburnation is the ivory-like, shiny patch on the medial femoral and tibial condyles. Prehistoric, California. One-half natural size.

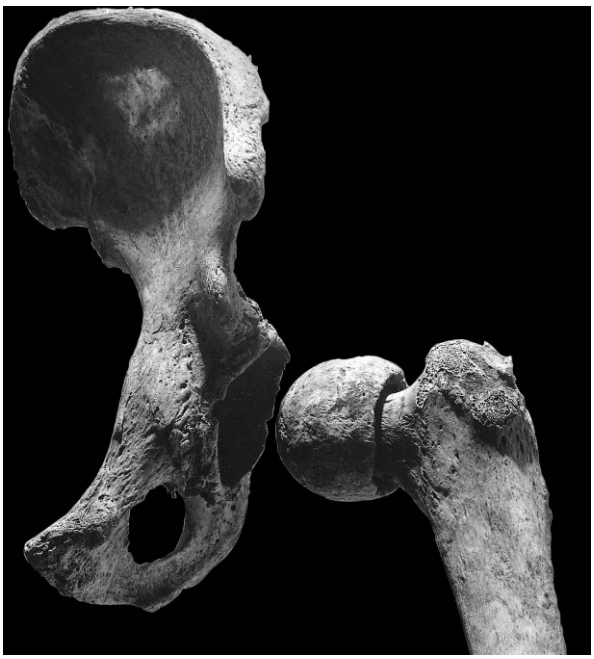

Figure 19.10 **Osteoarthritis.** Trauma to this left femur produced secondary osteoarthritic changes to the joint, seen here in the form of a bony extension and deformation of the femoral head and a buildup of osteophytes around the perimeter of the acetabulum. Prehistoric, California. One-third natural size.

19.5.2 Diffuse Idiopathic Skeletal Hyperostosis (DISH)

Diffuse idiopathic skeletal hyperostosis (DISH), while not a true joint disease — it doesn't involve any of the intervertebral synovial joints — is usually considered together with joint diseases. DISH involves the ossification of the lateral fibers of the anterior longitudinal spinal ligament. The result is smooth-surfaced bony bridges between adjacent vertebrae that create a "dripping candle wax" appearance. To be considered as DISH, these bony bridges must span at least 4 adjacent vertebrae on their anterolateral aspects. Unlike ankylosing spondylitis (see Section 19.5.4), DISH affects the anterior portions of vertebral bodies and leaves the intervertebral disk spaces and articular facets unaffected.

19.5.3 Rheumatoid Arthritis

Middle-aged women have a predisposition for this arthritis. Its exact cause remains unknown but almost certainly varies with genetic background. In rheumatoid arthritis, the immune system of the body attacks its own cartilage. Bone changes are atrophic and are especially focused in the hands and feet. The lesions are usually bilaterally symmetrical. Rheumatoid arthritis is the least common arthropathy in archaeological skeletal material. Woods and Rothschild (1988) argue that it is evidenced in New World skeletal remains, and Rothschild et al. (1990) propose recognition criteria for skeletal remains.

19.5.4 Ankylosing Spondylitis

An **ankylosis** is an abnormal immobility or fixation of a joint resulting from pathological changes in the joint. **Ankylosing spondylitis** (also called rheumatoid arthritis of the spine) is a chronic and usually progressive disease that affects the vertebral column. Ankylosing spondylitis begins in the region of T-12/L-1 and involves the ossification of the periphery of the annulus fibrosis, forming symmetric **syndesmophytes** (a bony outgrowth at the margin of a joint) between adjacent vertebrae, leading to the eventual immobilization of the intervertebral joints. Ortner (2003) notes that smooth ankylosis of the sacroiliac joint is common, as are syndesmophytes at the articular facets and costovertebral facets. Onset of ankylosing spondylitis can occur at any time from childhood onwards, but usually occurs between 20 and 40 years (Feldkeller et al., 2003).

19.6 Infectious Diseases and Associated Manifestations

Infectious disease has long been a major cause of death in human populations. Unfortunately, dealing with osteological evidence of infectious disease can be frustrating because few infectious diseases leave any direct evidence of their existence in the skeleton. Many of the chronic infectious diseases that *do* leave osteological signs produce morphologically overlapping responses, making differential diagnosis impossible.

Osteitis is a general term for an inflammation of bone tissue caused by infection or injury and is not specific as to cause. The terms **osteomyelitis** and **periostitis** are slightly more confusing because they serve to generally describe osteological conditions as well as to identify specific diseases. Periostitis is a symptom in disease syndromes such as syphilis, but it is also a common condition that can be result from many other diseases, including some that are not infectious diseases (Ortner, 2003). Because of their non-specific nature, caution should be exercised in diagnosing periosteal lesions on incomplete archaeological skeletons (Weston, 2008, 2009).

19.6.1 Bacterial Infections

There are many bacterial infections which, if left untreated for a sufficiently long period of time, will leave evidence of the infection on skeletal remains. Among the bacterial infections that can be diagnosed from skeletal remains are tuberculosis, leprosy, treponematosis (includes yaws and venereal syphilis), osteomyelitis, pneumonia, salmonellosis, brucellosis, actinomycosis, nocardiosis, cholera, and plague.

a. **Tuberculosis**. Tuberculosis is a chronic infectious disease that results from a bacterium, *Mycobacterium tuberculosis*. Infection is usually via the respiratory system, but other body parts, including bones, can also be affected. Bone and joint destruction can result from the infection. The presence of pre-Columbian tuberculosis in the New World is assessed by Buikstra (1981b) and Roberts and Buikstra (2003). Molecular work by Salo and colleagues confirmed osteological observations in 1994. A variety of bones can be affected by tuberculosis, but the vertebral column is the most common primary focus. The most common manifestation (when viewed from the side) is the collapse of one or several vertebral bodies causing a sharp angle in the spine (**kyphosis**). Differential diagnoses from osteomyelitis and septic arthritis are often possible because tuberculosis shows destruction and cavitation in cancellous bone, without extensive associated reactive bone. The pattern of element involvement, with the vertebrae and os coxae as foci, marks tuberculosis. In addition, there is no evidence of sequestration, an involucrum, or fusion of the joints. Ortner and Putschar (1981) discuss and illustrate the skeletal effects of tuberculosis.

b. **Treponemal Infections**. Skeletally significant diseases caused by a microorganism known as a spirochete (in the genus *Treponema*) are yaws (*T. pallidum pertenue*), endemic syphilis (*T. pallidum endemicum*), and venereal syphilis (*T. pallidum pallidum*). These diseases have a worldwide distribution today, but the decades-long controversy over their origins and original distribution is still not settled. Was syphilis (misdiagnosed as leprosy) or other treponemal disease present in the New World before the arrival of Columbus? Did treponemal diseases exist in the Old World before Columbus returned from the New World? Did they exist in both Old and New Worlds prior to 1492? The controversy is well-reviewed by Steinbock (1976), Baker and Armelagos (1988), and Ortner (2003). Evidence for pre-Columbian treponemal infection in the New World is reviewed by Ortner (2003), who states that there are many examples of New World treponemal infection that probably predate European contact, but notes that the dating for none of these is certain because they derive from older, poorly controlled excavations. Recent research by von Hunnius et al. (2006) and Mays et al. (2003) has demonstrated that treponemal disease was almost certainly already present in the British Isles before Columbus' voyage. The scarcity of pre-Columbian Old World examples of treponemal disease (*e.g.*, Schultz et al., 2003; Cardy, 1997; Roberts, 1994; Pálfi et al., 1992; and Stirland, 1991b), has focused attention on Viking merchants as possible culprits in the spread of treponemal disease (Malakoff, 2000).

   In syphilis, the microorganisms enter the body through the skin or mucous membrane sites. Tertiary syphilitic skeletal lesions occur progressively, usually beginning between 2 and 10 years after infection. These can be complex, but there is usually an osteological focus of the disease in the frontal and parietals, the facial skeleton, and the tibiae. Individual lesions may not be distinguishable from some cancers, tuberculosis, or other infectious changes. Steinbock (1976), Aufderheide and Rodríguez-Martín (1998), Buckley and Dias (2002), and Ortner (2003) provide further details.

c. **Osteomyelitis**. Osteomyelitis is bone inflammation caused by bacteria that usually initially enter the bone via a wound. This disease mainly affects the long bones (Figure 19.11) and is defined as an infection that involves the medullary cavity. However, usage in the paleopathology literature has often been imprecise. Osteomyelitis is almost always

Figure 19.11 **Osteomyelitis.** The tibia on the left shows localized reactive bone and a cloaca, whereas that on the right shows the result of a more extensive reaction to the infection. Radiographs of the two bones show clear involvement of the medullary cavity. Prehistoric, California. One-half natural size.

caused by pus-producing microorganisms (90% of the time by *Staphylococcus aureus*) and is thus called **suppurative**, or **pyogenic osteomyelitis**. The microorganisms can reach the bone directly, as a result of injury at any age, or via the bloodstream (hematogenous osteomyelitis, most often found in children). Characteristic hard tissue manifestations include an **involucrum** of coarsely woven bone around the original long bone cortex and one or more openings for pus drainage called **cloacae** (or **fistulae**). The latter open through the involucrum.

d. **Periostitis**. Periostitis is a condition of inflammation of the periosteum caused by trauma or infection. It is not a disease; rather, it is a symptom of several diseases. See Mensforth et al. (1978) for a complete review of its various etiologies. These authors demonstrate an age-specific distribution of periosteal reactions that seems to coincide with — and be a response to — infectious disease in infants and children in the prehistoric Libben population. Periostitis involves only the outer (cortical) bone, without involvement of the marrow cavity (as in osteomyelitis). It can be acute or chronic and it occurs any time the inner surface of the periosteum reacts to insult by forming woven bone that sleeves the underlying cortical bone (Figure 19.12).

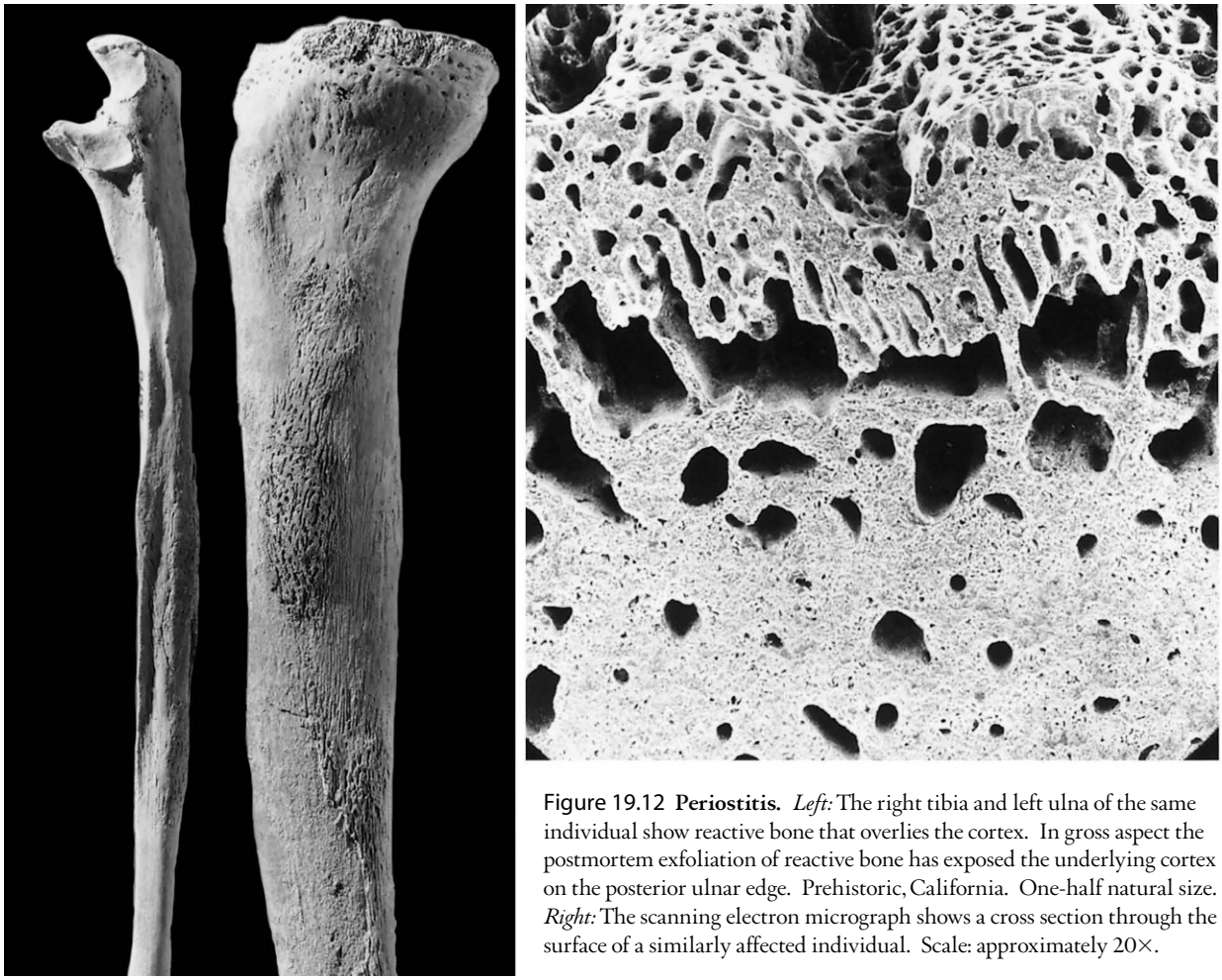

Figure 19.12 **Periostitis.** *Left:* The right tibia and left ulna of the same individual show reactive bone that overlies the cortex. In gross aspect the postmortem exfoliation of reactive bone has exposed the underlying cortex on the posterior ulnar edge. Prehistoric, California. One-half natural size. *Right:* The scanning electron micrograph shows a cross section through the surface of a similarly affected individual. Scale: approximately 20×.

19.6.2 Other Infections

In addition to bacterial infections, several other vectors of infection are known to leave evidence on skeletal remains. These include viral infections, fungal infections, and parasitic infections. Auferderheide and Rodríguez-Martín (1998) and Waldron (2009) provide extensive details on the skeletal manifestations of each of these types of infections.

19.7 Metabolic Diseases

Narrowly defined, metabolic disorders of bone are disorders in which a reduction in bone mass is the result of inadequate osteoid production, or mineralization or excessive deossification of bone. Nutritional deficiencies are usually classified under metabolic disorders. Hormonal disturbances can also lead to dramatic changes in normal skeletal anatomy.

19.7.1 Vitamin-D-related Syndromes

a. **Rickets**. Rickets is most often a nutritional disease resulting from an insufficient amount of vitamin D in the diet that causes a failure of mineral deposition in the bone tissue of children. As a result, excessive uncalcified osteoid accumulates, and the bone tissue remains soft and flexible. The disease was described in the 1600s, but its nutritional source was not discovered until the 1920s. The osteological effects of rickets are present throughout the skeleton, but they are most pronounced in the limbs, which are usually bent and distorted. The legs are characteristically bowed outward or inward.

b. **Osteomalacia**. The same lack of sufficient vitamin D in adults (due to either nutritional deficiency or intestinal malabsorption) is called osteomalacia, a disease usually linked to general malnutrition, particularly to deficiencies in protein, fat, calcium, and phosphorus. Osteomalacia is essentially "adult-onset rickets." The differences between osteomalacia and rickets are due to the fact that rickets is active during the period of skeletal growth, while osteomalacia acts on bones after they have finished growing. Osteomalacia's greatest effect is on bones in which remodeling is highest (ribs, sternum, vertebrae, and pelvis), making them subject to pathological fracture.

19.7.2 Scurvy

Scurvy is a metabolic disease caused by a long-term insufficient intake of vitamin C, which is essential for the production of collagen and, therefore, osteoid. Skeletal manifestations are most apparent in infants, usually in the form of cortical thinning and pathological fractures in rapidly growing bone areas. Maat (2004) provides a useful review and case study.

19.7.3 Osteoporosis

Osteoporosis, or **osteopenia** in the nonclinical situation, refers to the increased porosity (reduced density) of bone that is most often part of the aging process. It is a consequence of the failure of the organism to maintain the balance between bone resorption and formation. Postmenopausal women are most at risk for osteoporosis because of the cessation of estrogen production. Males are endowed initially with more bone mass than females and so do not become vulnerable to osteoporosis until later in life (in their 70s and 80s). The annual costs associated with osteoporosis in the United States alone are estimated at more than $19 billion (Burge et al., 2007).

Osteoporosis is associated with more than 2 million bone fractures in the elderly each year. Research into basic bone biology, particularly into the factors that activate and inactivate osteoclasts, has been spurred on by these statistics. Marcus et al. (2008) offer a comprehensive review of research on osteoporosis.

19.7.4 Poisoning

In addition to osteoporosis, scurvy, and vitamin-D-related syndromes, there are several kinds of intoxications and poisonings that are detectable on skeletal remains. See Aufderheide and Rodríguez-Martín (1998) for further details on these and other metabolic disorders that leave traces on skeletal remains.

19.8 Endocrine Disorders

The growth of the skeleton is controlled, in large part, by the secretion of hormones in the pituitary and thyroid. Pathology in these glands can lead to extreme skeletal changes.

19.8.1 Pituitary Disorders

a. **Gigantism**. Gigantism results from excessive production of somatotrophic hormone and consequent overstimulation of growth cartilages and gigantic proportions of the skeleton.
b. **Acromegaly**. Acromegaly is similarly caused by an overly productive pituitary, but after the epiphyses are fused. The most dramatic osteological manifestation of acromegaly is growth at the mandibular condyle and a resulting elongation and distortion of the lower jaw.
c. **Pituitary dwarfism**. Dwarfism is a general term for a variety of disorders that lead to greatly reduced stature and/or body proportions. Pituitary dwarfism results from an underactive pituitary gland, leading to reduced production of growth hormone and resulting in a normally proportioned but abnormally short individual.

19.8.2 Other Glandular Disorders

In addition to disorders of the pituitary gland, there are several other types of endocrine disorders. Disorders of the parathyroids, gonads, pancreas, thyroid, and adrenal glands also have effects on the skeleton. See Aufderheide and Rodríguez-Martín (1998) and Waldron (2009) for further details on these and other endocrine disorders that can be diagnosed on skeletal remains.

19.9 Hematopoietic and Hematological Disorders

19.9.1 Red Blood Cell Disorders (Anemias)

Anemias can affect the gross appearance of bones. Iron deficiencies, sickle cell anemia, and thalassemia all result in the expansion of spaces occupied by hematopoietic marrow within the bones. The result is often visible in the skull, with a widening of the diploë leading to a thickening of the cranial vault. Zimmerman and Kelley (1982) attempt a differential diagnosis of different types of anemias.

Porotic (or **spongy**) **hyperostosis** is a condition exhibiting lesions, usually of the cranial vault. These represent a thinning and often complete destruction of the outer table of the cranial vault that results in a sieve-like or "coral-like" appearance of the ectocranial surface. The lesions seem to be caused by anemia-associated hypertrophy of the diploë between the inner and the outer tables. Porotic hyperostosis is most often seen in immature individuals and is recognizable as a porosity of the cranial vault. It is usually bilaterally symmetrical, focused on the parietals and the anterolateral quadrant of the orbital roofs. The orbital lesions are called **cribra orbitalia**, and a similar disturbance of the endocranial surface is called **cribra cranii** (see Figure 19.13).

Causes of this bony reaction have been extensively speculated. Its high frequency in the southwest of North America was once thought to be because of a maize-based, iron-deficient diet. However, work on the phenomenon in a large sample of California skeletons (nonagricultural, fish-dependent) by Walker (1986) suggested that porotic hyperostosis is sometimes due to nutrient losses associated with diarrheal diseases rather than to diet *per se*, a position supported by Kent (1987). Walker et al. (2009) have recently argued that while porotic hyperostosis and cribra orbitalia may be associated with iron-deficiency anemia, they cannot be caused by it. Instead, they suggest that the majority of cases are caused by megaloblastic anemia acquired by infants through ingesting vitamin-B_{12}-depleted breast milk while suffering gastrointestinal infections. They also conclude that cribra orbitalia is likely to have many causes, including a co-deficiency of vitamins C and B_{12}. Wapler et al. (2004), in a histological examination of archaeological examples of cribra orbitalia, found evidence for multiple probable causes for this condition.

In a study of skeletal remains from the prehistoric southwest, Palkovich (1987) suggests that endemically inadequate maternal diet can combine with infection to produce very early onset of iron-deficient anemia with resultant porotic hyperostosis. Research by Stuart-Macadam (1987a, b) stresses the association between osteological manifestations and anemia, a subject further reviewed by Wapler et al. (2004). Using the prehistoric Libben sample, Mensforth et al. (1978) demonstrate that porotic hyperostosis is commonly caused by the normal sequelae of infection.

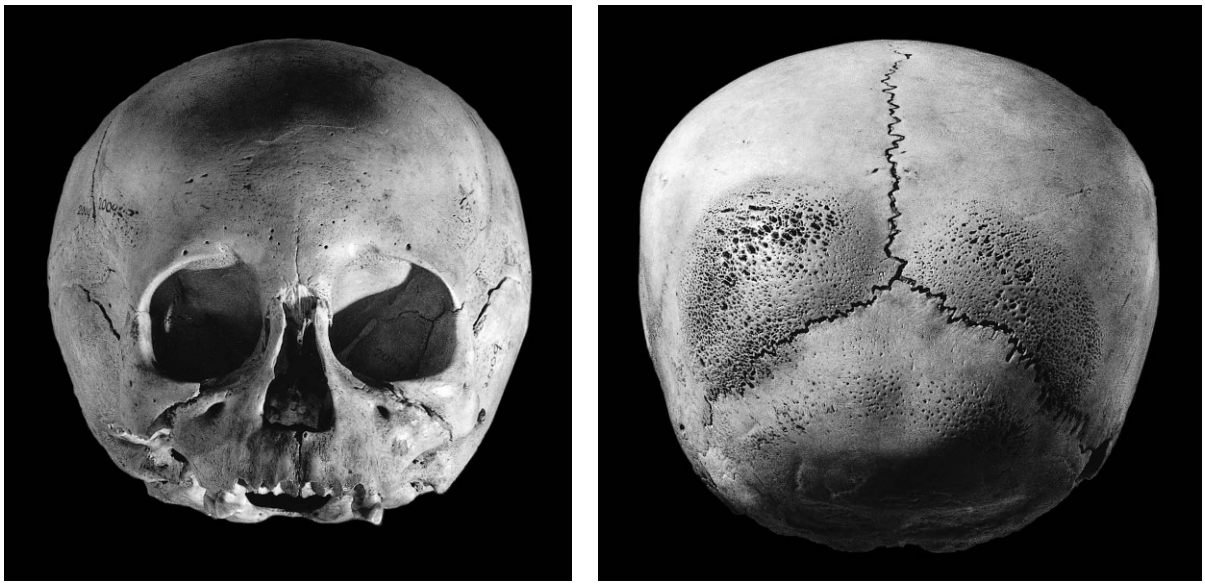

Figure 19.13 **Cribra orbitalia, porotic hyperostosis.** The skull of a 5-year-old child is shown in anteroinferior and posterior views to display the cribra orbitalia of the frontal (in the roofs of the orbits) and spongy hyperostosis of the parietals and occipital. Prehistoric, Peru. One-half natural size.

As noted earlier, they found a direct relationship between periostitis (infection) and porotic hyperostitis (iron deficiency) in subadults. This is consistent with clinical data that suggest that iron may be sequestered within the body as a defense against infection. Thus, much prehistoric porotic hyperostosis may be the secondary consequence of infectious disease, not of diet. Indeed, Stuart-Macadam (1991) posits that porotic hyperostosis is the result of an interaction among customs, diet, hygiene, parasites, and infectious diseases. She feels that evidence for chronic disease in skeletal material should not be interpreted as an individual's inability to adapt to the environment, but rather as evidence of the individual's fight for health against the pathogen.

19.9.2 White Blood Cell Disorders

a. **Multiple myelomas**. Multiple myeloma (MM) is a rare primary malignant tumor of hematopoietic tissue. Its effect on bone tissues is a widespread pattern of lytic lesions on various skeletal elements.
b. **Langerhans Cell Histiocytosis (Histiocytosis-X)**. Langerhans cells are histiocytes (macrophagocytic cells responsible for consuming dead or abnormal cells) that can travel throughout the body. There are three types of Langerhans cell histiocytosis (LCH), mostly seen in children under 10: unifocal LCH (eosinophilic granuloma), multifocal unisystem LCH (Hand-Schüller-Christian disease), and multifocal multisystem LCH (Letterer-Siwe disease). While the distribution of lesions differs, the bony lesions are similar for all forms of LCH. Destructive lesions of the cranial vault and orbit are the most common, with undulating, beveled margins and no reactive bone formation. Ortner (2003), Aufderheide and Rodríguez-Martín (1998), and Dorfman and Czerniak (1998) provide further diagnostic information.
c. **Leukemia**. Manifestations of leukemia can be difficult to differentiate from other hematological malignancies, osteomyelitis, and some infections, and diagnosis usually involves ruling these other diseases out. Rothschild et al. (1997) provide criteria by which Leukemia can be recognized in skeletal remains.

19.10 Skeletal Dysplasias

19.10.1 Achondroplasia

As stated earlier, dwarfism is caused by a variety of conditions. **Achondroplasia** is a hereditary form of dwarfism characterized by limb shortening, almost normal trunk and vault development, and a small face (Figure 19.14). It is a hereditary disease caused by the congenital disturbance of cartilage formation at the epiphyses, and constitutes a skeletal dysplasia rather than an endocrine disturbance (for example, pituitary dwarfism).

19.10.2 Osteogenesis Imperfecta

Osteogenesis imperfecta (OI or brittle bone disease) is the name of a group of pathological conditions characterized by the inability to produce type I collagen, which forms 90% of bone protein (Ortner, 2003). Because collagen forms the matrix into which hydroxyapatite is deposited, individuals with OI can have severely deformed bones prone to fracture. While OI can affect nearly any bone, the most commonly affected are the bones of the legs, the vertebrae, ribs, and clavicles (Aufderheide and Rodríguez-Martín, 1998).

Figure 19.14 **Achondroplasia.** The left humerus and left femur of an achondroplastic dwarf (*far left* and *far right*) are compared with a normal human humerus and femur from a second individual (*center*). Note the disproportions and fully adult status of the achondroplastic individual. Prehistoric, California. One-fourth natural size.

Figure 19.15 **Fibrodysplasia ossificans progressiva.** Advanced bony manifestations of FOP in a 39-year-old man. Courtesy of Fred Kaplan, Mütter Museum, College of Physicians of Philadephia (Shafritz et al., 1996).

19.10.3 Fibrodysplasia Ossificans Progressiva

Fibrodysplasia ossificans progressiva (FOP) is a heritable disorder of connective tissue characterized by congenital malformation of the large toes and progressive, disabling endochondral osteogenesis in predictable anatomical patterns. Disease progression brings fusion of adjacent bones of the spine, limbs, thorax, and skull, leading to immobilization (Figure 19.15). The abnormal bone buildup occurs because white blood cells erroneously manufacture BMP-3, triggering inappropriate heterotopic ("other" + "place") bone growth at sites of injury (Shafritz et al., 1996). See Section 3.9 for further details on the specific metabolic processes involved.

19.10.4 Other Skeletal Dysplasias

The number of named skeletal dysplasias has increased greatly in recent years, due primarily to research in genetics and biochemistry. The most recent classification of skeletal disorders lists 372 distinct dysplasias, with the causative gene identified for nearly 60% (215) of these (Superti-Furga et al., 2007). No single volume in paleopathology has kept up with the pace of this new research, but the most comprehensive reference available is Chapter 12 of Aufderheide and Rodríguez-Martín (1998). An authoritative source for information about disorders with a genetic component is the Online Mendelian Inheritance in Man database (OMIM, n.d.). The online version of the classification of disorders (*http://www.isds.ch/Nosology2006.html*) has links to the relevant OMIM entries.

19.11 Neoplastic Conditions

Neoplasms (or tumors) are localized areas of unregulated cell growth. Neoplastic growths that remain small and localized because they are unable to destroy surrounding tissues or migrate to other areas of the body are called benign growths. Neoplastic growths that can destroy nearby tissues and that can migrate throughout the body and form new growth centers (metastases) are called malignant growths, or cancers. Modern classifications of skeletal tumors name over 40 different tumor types in bone and associated cartilage and fibrous connective tissue. Histological specimens and biochemical data are usually needed to sort these out.

Tumors are classified according to the tissues in which they originate. For example, while it creates a widespread pattern of lytic lesions across multiple skeletal elements, multiple myeloma is considered a primary malignant tumor of hematopoietic tissue. While some tumors found on skeletal remains originate from bone tissue, skeletal tumors usually stem from other tissue sources, and their appearance can be very dramatic.

19.11.1 Osteomata (Osteomas)

An **osteoma** (plural: *osteomata* or *osteomas*) is a solitary, well-defined mound of compact bone, usually less than 10 mm in diameter (Aufderheide and Rodríguez-Martín, 1998). They are benign tumors and are most often found on the cranium and face, but have also been reported on the femur, tibia, humerus, and clavicle. Osteomas on the ectocranial surface of the cranial vault are often called "button" osteomas (Eshed et al., 2002). Button osteomas are hard, dense, and ivory-like in appearance and occur in about 1% of all people. **Auditory exostoses** are osteomata of the inner aspect of the external acoustic meatus.

19.11.2 Primary Malignant Bone Tumors

Primary malignant bone tumors (tumors originating from bones) are uncommon ($<$ 1% of all malignant tumors: Waldron, 2009), and are much less common than metastatic bone tumors (tumors that originate elsewhere and then spread to bone). There are three types of primary malignant bone tumors: osteosarcomas, chondrosarcomas, and Ewing's sarcomas.

a. **Osteosarcomas** (**osteogenic sarcomas**). Osteosarcoma is the most common primary malignant bone cancer affecting younger individuals, in both modern (A.D.A.M., 2010) and ancient (Aufderheide and Rodríguez-Martín, 1998) populations. The most commonly formed osteosarcomas occur during the growth period, affecting individuals aged 10

to 25 years (A.D.A.M., 2010). Osteosarcoma tends to occur in the distal femur, proximal tibia, and proximal humerus (Ortner, 2003), but has been noted in other bones, such as the mandible (Aufderheide and Rodríguez-Martín, 1998). Osteosarcomas begin inside the bone in the metaphysis, and spread outwards and into the medullary cavity. Osteosarcomas rarely affect the epiphyses or joint surfaces. Depending on the stage and progression of the tumor, lesions may be primarily lytic (Ortner, 2003), or they may present either a layered ("onion skin") or a radial ("sunburst") pattern of involvement once beyond the periosteum (Aufderheide and Rodríguez-Martín, 1998).

b. **Chondrosarcomas**. Chondrosarcomas originate in cartilage, and typically affect older adults (Aufderheide and Rodríguez-Martín, 1998). The most common locations for chondrosarcomas are the os coxae, femur, humerus, ribs, and scapula.

c. **Ewing's sarcomas**. Ewing's sarcoma begins in the medullary cavity and affects men twice as often as women. The tumors typically occur in childhood, but can occur at any point in life. The most common locations for Ewing's sarcomas are the femur, tibia, humerus, and os coxa. In contrast to osteosarcoma, Ewing's sarcomas typically develop in the middle of the long bones. Ewing's sarcomas usually present a perpendicular ("hair on end") or a layered ("onion skin") pattern of involvement once beyond the periosteum (Waldron, 2009; Aufderheide and Rodríguez-Martín, 1998).

Waldron (2009), Ortner (2003), Aufderheide and Rodríguez-Martín (1998), and Steinbock (1976) all provide additional information on these rare cancers.

19.11.3 Osteochondromas

The most common tumor of bone and cartilage is an **osteochondroma** (also called an **osteocartilagenous exostosis**). These are benign tumors that are usually asymptomatic. They always arise at epiphyseal lines and protrude at right angles to the long axis of a bone. They can superficially resemble ossified tendons in many cases, but they have a core of trabecular bone that is continuous with the trabecular bone of the metaphysis (Aufderheide and Rodríguez-Martín, 1998). Most develop before 30 years of age, and while they can occur near the metaphysis of any long bone, the most common locations are the distal femur and proximal tibia.

19.11.4 Fibrosarcomas (Fibroblastic Sarcomas)

Fibrosarcomas, like osteosarcomas, begin inside the bone in the metaphysis and spread into the medullary cavity. Unlike osteosarcomas, fibrosarcomas are usually confined to the central portions of the bone, although they do spread beyond the periosteum (Waldron, 2009). Fibrosarcomas typically occur in adults aged 30 to 60 years, and both sexes are equally afflicted (Aufderheide and Rodríguez-Martín, 1998). Fibrosarcomas most often occur in the femur and tibia, followed by the humerus and os coxae; tumors have also been reported in the mandible and maxilla (Waldron, 2009).

19.11.5 Other Neoplastic Conditions

In addition to those listed above, there are several other types of neoplastic conditions affecting bone. See Waldron (2009), Ortner (2003), and Aufderheide and Rodríguez-Martín (1998) for fuller coverage of other benign and malignant tumors that can be found on skeletal remains.

19.12 Diseases of the Dentition

Because the teeth interact directly with the environment, they are susceptible to damage from physical and biological influences not operating on other skeletal elements. An examination of dental pathology can be useful for investigating the health and diets of individuals and populations. Even though tooth wear that is excessive by today's standards has characterized humans and their ancestors for millions of years, it should be noted that tooth wear and artificial tooth modification have been included in this chapter only for convenience. Lukacs (1989) divides dental diseases into four categories: infectious, degenerative, developmental, and genetic. All of these, of course, are interrelated in the dental health of the individual. Tooth wear should be thought of as a pathology only when it is so extreme that the associated bone is affected negatively. Hillson (2002) provides a useful review of dental pathology, with guidelines for assessing and scoring caries and periodontal disease (following his 2001 recommendations). Chapter 18 considers how tooth wear (dental macrowear) may be used to age skeletal material and discusses how such wear can be used to assess prehistoric diets. Figure 19.16 shows how extreme dental macrowear was under many prehistoric conditions.

19.12.1 Periodontal Disease

Periodontitis is the inflammation of tissues around a tooth. It can involve both soft tissues and the bone itself. Periodontal disease in skeletal remains is recognized as a result of infection of the alveolar bone and adjacent tissues. It causes recession of the alveolar bone, as either a horizontal lowering of the crest of the alveolar process or as an irregular lowering of the process, with pockets or wells expanding into the cancellous bone of the jaws. The agents of infection are microorganisms, and the disease is usually due to the combined effects of large, mixed communities of bacteria. An **abscess** is a localized collection of pus in a cavity formed by tissue disintegration. Abscesses are often found as cavities within alveolar bone near the tooth root apices. Figure 19.17 illustrates these features. Clarke and Hirsch (1991) provide a thorough account of factors influencing alveolar bone. Oztunc et al. (2006) question the assumption of low incidence of periodontal disease in archaeological populations. Eshed et al. (2006) examine measures of dental health and wear before and after the transition to agriculture in the Near East, concluding that the differences demonstrate more of a gradient than a dichotomy.

Figure 19.16 **Tooth wear.** Heavy attrition on dentition of this adult male has eliminated all but the third molar crowns. The individual continued to chew on the stubs of his incisors, canines, premolars, and second molar after the tooth crowns had worn away. Such wear is a normal phenomenon in older individuals from aboriginal populations with grit in the diet. Prehistoric, California. Natural size.

19.12.2 Caries

Dental **caries** is a disease process characterized by the progressive decalcification of enamel or dentine. The macroscopic appearance of caries can vary from opaque spots on the crown to gaping cavities in the tooth. A prerequisite for the formation of dental caries is **dental plaque** and a diet that includes fermentable carbohydrates. Plaque is the matrix and its inhabiting community of bacteria that forms on the tooth. Carious lesions can begin anywhere that plaque accumulates, most often in the fissures of tooth crowns and in the interproximal areas. Figure 19.17 shows a large carious lesion.

Larsen (1983) observed that the prehistoric shift to agriculture on the Georgia coast led to an increase in the frequency of carious lesions, most markedly in females. This finding indicated differing subsistence roles between the sexes and is an example of how archaeological and osteological data can be combined in insightful ways. Walker and Erlandson (1986) saw a similar but inverse shift on the California coast. Here, prehistoric people made a subsistence shift from a cariogenic diet, consisting mostly of plant foods, to an intensive exploitation of fish.

Lanfranco and Eggers (2010) recommend a system for recording the specific location and depth of carious lesions, to be evaluated together with the level of dental macrowear. Lukacs (2008) links the higher incidence of caries in women at the advent of agriculture to pregnancy-associated hormonal and dietary changes, combined with an increase in fertility.

19.12.3 Enamel Hypoplasia

Enamel hypoplasia is a condition characterized by transverse lines, pits, and grooves on the surface of tooth crowns. These disturbances are defects in enamel development. Amelogenesis, or enamel formation, begins at the occlusal apex of each tooth crown and proceeds rootward, ending where the crown meets the root at the cervicoenamel line. During this process, stress to the organism may result in a temporary upset of ameloblastic activity and a consequent enamel defect marking the interruption of development. These enamel hypoplastic defects can take many forms, ranging from single pits to lines to grooves. As Danforth et al. (1993) note, because scoring of these features varies greatly between observers, interobserver and intraobserver bias must be considered in any comparison of results from different studies.

Figure 19.17 **Caries and abscess.** Bilateral carious lesions of the first molars are evident on this individual. A related abscess around the distal first molar root is seen posterosuperior to the right mental foramen. Prehistoric, California. Natural size.

Several different factors can cause dental hypoplasias (see Goodman and Armelagos (1985), and Skinner and Goodman (1992), for reviews), all of them the result of metabolic insult to the organism. Like Harris lines, hypoplastic bands in individuals can indicate the age at insult. Study of these developmental defects in populations can give insights into patterns of dietary and disease stress in prehistoric groups. Goodman and Rose (1991) provide a fine discussion of enamel hypoplasias as clues about the adequacy of prehistoric diet, and Goodman and Song (1999) provide more detail on estimating ages of hypoplastic defect formation. Figure 19.18 shows heavy hypoplastic banding and pitting on a child's permanent canines.

19.12.4 Dental Calculus

Dental calculus is mineralized plaque on a tooth surface. The fact that calculus can trap food debris has been used by Dobney and Brothwell (1986) as an approach for ascertaining aspects of prehistoric diet. Figure 19.18 shows remnants of calculus buildup.

19.12.5 Artificial Dental Modification

A variety of cultural practices (technically, forms of trauma) impact dentition. People engrave, color, and even intentionally pull out (**evulse**) teeth for cosmetic purposes. Brothwell (1981) illustrates the variety of decorations encountered on dental remains, primarily on the anterior teeth. These artificial incisions should not be confused with hypoplasias. Milner and Larsen (1991) review filing, chipping, inlays, and other alterations, referring to these as **dental mutilations**, whereas purposeful removal of usually anterior teeth is termed **ablation**.

Frayer (1991) and many others have interpreted the interproximal grooves sometimes present between adjacent teeth as grooves left by the use of toothpicks, and such features are even known from fossil hominid teeth of more than two million years in age. Hlusko (2003) infers that these grooves were made by grass stalks used as toothpicks. Fox (1992) reviews literature on cultural striations in the human dentition, noting several studies that have shown handedness based on the directionality of oblique scratch marks on the labial surfaces of upper incisors, particularly among paleolithic hominids. These marks were presumably made because food was secured by using the front teeth as a vise while pieces were cut away with a stone implement that came into contact with the teeth.

Figure 19.18 **Enamel hypoplasia.** This 12-year-old child suffered a metabolic upset during the formation of his canines (to see when this occurred, consult Section 18.3.1). This upset recorded itself in the linear hypoplasia seen bilaterally on the labial surface of the canine crowns. Calculus is also seen on the labial surface of the incisor crowns. Prehistoric, California. Natural size.

19.13 Musculoskeletal Stress Markers

In forensic and archaeological contexts, it is important to determine the occupational and social status of individuals now represented by skeletons. It may be possible to gain insight into the activities habitually performed by an individual by examining the patterns of musculoskeletal stress markers on that individual. While musculoskeletal stress markers can only provide basic information on the relative loading of the various muscles on the skeleton, researchers have linked patterns of stress markers to specific activities and by inference, to specific occupations. As a result, musculoskeletal stress markers are often called "occupational markers." The inferences drawn range from the straightforward, such as determining handedness (Steele, 2000), to the ambiguous, such as fruit-pickers' cervical spine or dog-walkers' elbow (Capasso et al., 1998). Kennedy (1989) traces the history of occupational studies in anthropology and medicine. Capasso et al. (1998) examine occupational markers from a functional morphological perspective. Villote et al. (2010) review the evidence for occupational stress markers on the upper limb, and Weiss (2004) similarly reviews the evidence for markers on the lower limb. Waldron's (1994) call for critical evaluation of claims is apt and enlightening. He notes that in their fervor to deduce as much as possible about the lives of ancient individuals, osteologists have made some extravagant claims about environmental stresses, parity, social status, and occupation. As he notes, some authors have been unable to resist the temptation to deduce an underlying cause for every bony lesion, identifying individuals as horsemen, sling throwers, weavers, and corn grinders based on the presence of bowed limb bones, spinal deformities, and osteoarthritis at various joints.

Perhaps because information regarding occupation and status is so valuable in an archaeological context, not enough attention is paid to the premises that underlie such identifications. Waldron's (1994) examination of whether patterns of osteoarthritis can be used to identify occupation is a useful departure. Proceeding from known to unknown, he notes that there is a wealth of modern clinical data on osteoarthritis and occupation. He finds no convincing epidemiological evidence of a consistent, coherent relationship between a particular occupation and a particular form of osteoarthritis. Indeed, given the fact that sex, race, weight, movement, and genetic predisposition are all known factors that influence development of the disease, this is not surprising. Waldron (1994) concludes that because we know that occupation is not the sole cause of osteoarthritis, there cannot be any likelihood of being able to deduce the former from the latter. Furthermore, even in cases in which occupationally related activity does seem to be important in determining the expression of arthritis, there are no unique features about this expression — most people who develop arthritis at the finger joints are not mill workers, even though mill workers do develop arthritis at these joints. For single skeletons, therefore, the prospects seem bleak.

On a populational basis, however, the prospects are better. Here, by examining the patterns of osteoarthritis in each skeleton, and among all skeletons, it might be possible to draw conclusions about activity pattern differences on a populational level. This is the underlying basis of the only comprehensive skeletal study of activity-induced pathology, a small populational study of Canadian Inuits in which activity patterns such as kayaking, harpoon throwing, and sewing were related to osteoarthritic patterns on a populational basis (Merbs, 1983). A study by Stirland (1991a) on sailors from King Henry VIII's A.D. 1545 flagship, *Mary Rose*, is another, smaller, and more limited sample in which an attempt to relate a paleopathological pattern to occupation is made. Even in the best of cases, however, it remains impossible to definitively conclude that any single individual had a particular occupation based on any particular arthritic joint in his or her skeleton. As Waldron (1994: 98) cautions, "There is a perfectly understandable drive to make the most of what little evidence survives in the skeleton and this sometimes has the effect of overwhelming the critical faculties." The osteologist should be wary of poorly supported claims about diet, disease, demography, and occupation at both the level of the individual skeleton and the level of the population sample.

Suggested Further Readings

Aufderheide, A. C., and Rodríguez-Martín, C. (1998) *The Cambridge encyclopedia of human paleopathology.* Cambridge, UK: Cambridge University Press. 496 pp.

Detailed descriptions and abundant illustrations make this an invaluable reference.

Brickley, M., and Ives, R. (2008) *The bioarchaeology of metabolic bone disease.* Boston, MA: Elsevier/Academic Press. 333 pp.

A comprehensive examination of chemical and age-related bone pathology in paleontological and modern contexts.

Cohen, M. N., and Crane-Kramer, G. M. M. (2007) *Ancient health: Skeletal indicators of agricultural and economic intensification.* Gainesville, FL: University Press of Florida. 432 pp.

This selection of essays traces the osteological effects of technological and economic advances of the late Holocene.

Mann, R. W., and Hunt, D. R. (2005) *Photographic regional atlas of bone disease: A guide to pathologic and normal variation in the human skeleton.* Springfield, IL: C. C. Thomas. 297 pp.

An excellent visual resource delineating normal variation and pathology, this revised edition replaces drawings from past editions with images.

Ortner, D. J. (2003) *Identification of pathological conditions in human skeletal remains* (2nd ed.). San Francisco, CA: Elsevier/Academic Press. 645 pp.

An outstanding text; essential for any work in paleopathology.

Pinhasi, R., and Mays, S. (Eds.) (2008) *Advances in human palaeopathology.* Hoboken, NJ: John Wiley and Sons. 408 pp.

This text presents multi-disciplinary, cutting-edge methods in the field of paleopathology.

Powell, M. L., and Cook, D. C. (2005) *The myth of syphilis: The natural history of treponematosis in North America.* Gainesville, FL: University Press of Florida. 509 pp.

This selection of historical and archaeological essays follows the North American history of endemic and epidemic treponemal infection.

Roberts, C., and Manchester, K. (2007) *The archaeology of disease* (3rd ed.) Ithaca, NY: Cornell University Press. 338 pp.

This revised edition updates its summary of various methods of identifying disease in the archaeological record.

Steinbock, R. T. (1976) *Paleopathological diagnosis and interpretation.* Springfield, IL: C. C. Thomas. 423 pp.

A well-illustrated classic text on paleopathology.

Tyson, R. A. (Ed.) (1997) *Human paleopathology and related subjects: An international bibliography.* San Diego, CA: San Diego Museum of Man. 716 pp.

A guide to the paleopathological literature.

Waldron, T. (2007) *Palaeoepidemiology: The epidemiology of human remains.* Walnut Creek, CA: Left Coast Press. 148 pp.

This volume details quantitative methods employed in paleoepidemiological research and includes advice on designing paleoepidemiological studies.

Waldron, T. (2009) *Palaeopathology (Cambridge manuals in archaeology).* New York, NY: Cambridge University Press. 279 pp.

Primarily method-oriented in its approach, this book recommends a standardized program of paleopathological diagnosis.